

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 16:15:03 ON 01 APR 2004

L1 146480 S ATHEROSCLEROSIS
L2 6969337 S PREVENT? OR TREAT?
L3 43817 S L1 AND L2
L4 1879104 S REVIEW?
L5 6802 S L3 AND L4
L6 8 S AMINOISOINDOLINE
L7 0 S L1 AND L6
L8 2819439 S HEART OR KIDNEY
L9 0 S L8 AND L6
L10 3 S L2 AND L6
L11 3 DUP REM L10 (0 DUPLICATES REMOVED)
L12 1227 S INDOLINE?
L13 0 S L5 AND L12
L14 2431 S ?INDOLINE
L15 0 S L14 AND L5
L16 71456 S TABLET?
L17 12 S L16 AND L5
L18 11 DUP REM L17 (1 DUPLICATE REMOVED)

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 DOCUMENT NUMBER: PubMed ID: 12215067
 TITLE: Micronised fenofibrate: an updated **review** of its clinical efficacy in the management of dyslipidaemia.
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AB Micronised fenofibrate is a synthetic phenoxy-isobutyric acid derivative (fibric acid derivative) indicated for the **treatment** of dyslipidaemia. Recently, a new **tablet** formulation of micronised fenofibrate has become available with greater bioavailability than the older capsule formulation. The micronised fenofibrate 160mg **tablet** is bioequivalent to the 200mg capsule. The lipid-modifying profile of micronised fenofibrate 160mg (**tablet**) or 200mg (capsule) once daily is characterised by a decrease in low-density lipoprotein cholesterol (LDL-C) and total cholesterol (TC) levels, a marked reduction in plasma triglyceride (TG) levels and an increase in high-density lipoprotein cholesterol (HDL-C) levels. Micronised fenofibrate 200mg (capsule) once daily produced greater improvements in TG and, generally, in HDL-C levels than the hydroxymethylglutaryl coenzyme A reductase inhibitors simvastatin 10 or 20 mg/day, pravastatin 20 mg/day or atorvastatin 10 or 40 mg/day. Combination therapy with micronised fenofibrate 200mg (capsule) once daily plus fluvastatin 20 or 40 mg/day or atorvastatin 40 mg/day was associated with greater reductions from baseline than micronised fenofibrate alone in TC and LDL-C levels. Similar or greater changes in HDL-C and TG levels were seen in combination therapy, compared with monotherapy, recipients. Micronised fenofibrate 200mg (capsule) once daily was associated with significantly greater improvements from baseline in TC, LDL-C, HDL-C and TG levels than placebo in patients with type 2 diabetes mellitus enrolled in the double-blind, randomised Diabetes **Atherosclerosis** Intervention Study (DAIS) [$>$ or \approx 3 years follow-up]. Moreover, angiography showed micronised fenofibrate was associated with significantly less progression of coronary **atherosclerosis** than placebo. Micronised fenofibrate has also shown efficacy in patients with metabolic syndrome, patients with HIV infection and protease inhibitor-induced hypertriglyceridaemia and patients with dyslipidaemia secondary to heart transplantation. Micronised fenofibrate was generally well tolerated in clinical trials. The results of a large (n = 9884) 12-week study indicated that gastrointestinal disorders are the most frequent adverse events associated with micronised fenofibrate therapy. Elevations in serum transaminase and creatine phosphokinase levels have been reported rarely with micronised fenofibrate. In conclusion, micronised fenofibrate improves lipid levels in patients with primary dyslipidaemia; the drug has particular efficacy with regards to reducing TG levels and raising HDL-C levels. Micronised fenofibrate is also effective in diabetic dyslipidaemia; as well as improving lipid levels, the drug reduced progression of coronary **atherosclerosis** in patients with type 2 diabetes mellitus. The results of large ongoing studies (e.g. FIELD with approximately 10 000 patients) will clarify whether the beneficial lipid-modifying effects of micronised fenofibrate result in a reduction in cardiovascular morbidity

and mortality.

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ACCESSION NUMBER: 2003184094 EMBASE

TITLE: **Treating** dyslipidemic patients with
lipid-modifying and combination therapies.

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SOURCE: Pharmacotherapy, (1 May 2003) 23/5 (625-637).

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FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery

030 Pharmacology

037 Drug Literature Index

038 Adverse Reactions Titles

039 Pharmacy

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Updated guidelines from the National Cholesterol Education Program give greater emphasis to lipoproteins other than low-density lipoprotein cholesterol (LDL) than previous guidelines. Although statins remain first-line therapy for most patients to lower LDL, combination therapy is the next logical step in achieving goals in patients with mixed dyslipidemia or elevated LDL despite statin therapy. As the prevalence of diabetes, metabolic syndrome, and atherogenic dyslipidemia rises, the importance of **treating** the total lipid profile becomes even more crucial. Niacin, fibrates, and bile acid sequestrants are effective in combination with statins in lowering LDL, triglycerides, and total cholesterol levels and increasing high-density lipoprotein cholesterol (HDL). Although combination therapies may increase the risk of myopathy, both fibrate-statin and niacin-statin combinations are considered safe. In addition, niacin-statin therapy reduces atherosclerotic progression and coronary events. New pharmacologic formulations exist that will further affect **treatment**: a single-tablet combination of lovastatin and extended-release niacin is available, as is ezetimibe, a cholesterol-absorption inhibitor. In all, both HDL and triglyceride levels correlate with cardiovascular risk and should be considered secondary targets of therapy. Combination therapy can be safe and effective and can be constructed to affect all lipoprotein parameters.

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ACCESSION NUMBER: 2003401969 EMBASE

TITLE: HMG-CoA reductase inhibitors - A **review** of the
recent patent literature.

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SOURCE: IDrugs, (2002) 5/3 (266-277).

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LANGUAGE: English

SUMMARY LANGUAGE: English

AB Statins are very potent inhibitors of HMG-CoA reductase, the rate-limiting enzyme in cholesterol biosynthesis at the mevalonate level. Today there is an increasing tendency to **treat** hypercholesterolemia aggressively, hence, the greater use of statins worldwide. The pleiotropic effect of statins is well documented. Examination of the patent literature reveals that in the past year pharmaceutical companies continued to be very active in this area. Accumulated knowledge of the actions of statins shows that they may be involved in many more processes than originally anticipated. Hence, in addition to 'old' indications (hypercholesterolemia, hyperlipidemia and **atherosclerosis**) many patent applications published in 2001 attempted to cover combination therapies, widening indications for statins to almost all known diseases. Many of the 'new' claims are not well substantiated and biological data are absent. Based on the magnitude of cardiovascular disease and aging population globally this area of drug discovery will continue to be an important area of research for all pharmaceutical companies. .COPYRGT. PharmaPress Ltd.